Endovascular Management of Intradural Berry Aneurysms

Review of 203 Consecutive Patients Managed between1993 and 1998

Morphological and Clinical Results at Mid-Term Follow-up

A. FUSE, G. RODESCH, H. ALVAREZ, P. LASJAUNIAS

Service de Neuroradiologie Diagnostique et Thérapeutique, Hôpital de Bicêtre, Le Kremlin Bicêtre; France

Summary

Endovascular management of intracranial arterial aneurysms (AA) is well described and performed by many teams. The aim of this work is to review a series of consecutive cases treated in our institution and to compare to the data available in the literature.

225 AA were seen in Bicêtre between 1993 and 1998 in 203 patients. 201 of them (in 180 patients) were treated by our group. The endovascular treatment, its indications, results and complications have been reviewed and studied. The clinical follow-up of the patients has been evaluated.

A female dominance was noted (64.5%) with a mean age of patients of 44.3 years. 65.6% of patients were treated in the acute phase after intracranial haemorrhage, 72% of them being Hunt and Hess grade 1 or 2. Most of these AA (73.6%) were located in the anterior circulation. In 86.1% of cases the AA was smaller than 10 mm. 85.6% of the AA needed only one session of endovascular therapy. No mortality occurred in the group of unruptured AA. Overall management mortality was 11% in ruptured AA (3.5% in HH1-2, 30.3% in HH3-5). Technical or transient complications occured in 11.6% of cases, but permanent morbidity was seen in 3.1% of cases. Control angiograms were performed 3 months and one year after therapy. In doubtful cases a control at 6 months was also performed. 100% occlusion rate was noted in 60.8% of cases; 22.8% of AA were occluded between 90-99%, and 13.3% between 80-90%. Only 3.1% of AA had an occlusion rate of less than 80%. One patient with a ruptured basilar tip AA which was partially coiled regrew and rebled three months after. The patient declined the recommended complementary surgery. Clinical follow up of patients with ruptured AA treated by embolisation shows satisfactory results with 8.,5% of GOS 1-2, 3.4% of GOS 3-4, and 11% of GOS 5 (mortality).

Overpacking of the AA may not be necessary to protect patients from (re)bleeds over time. The related technical risks and increased costs of dense overpacking do not seem justified. Secondary thrombosis of the ruptured AA after coiling is more often seen than coil compaction. Analysis of the AA architecture and recognition of false aneurysms are mandatory in order to obtain good clinico-morphological logical results.

Introduction

The most widely accepted treatment for intracranial aneurysms is still for many teams surgical clipping, which has proven its efficacy over time. Embolisation has tried very early to offer an alternative to surgery, but balloons [7,14] and microcoils [5] have not fulfilled the main

therapeutic objective required, which is the protection of the patient from hemorrhage. It is less than ten years ago that Guglielmi detachable coils (GDC; Target Therapeutics, Fremont, Calif.) [10,11] were introduced. They are currently considered to be the most efficient embolisation technique for AA, allowing selective exclusion of the sac by controlled placement of electrolytically detached platinum coils with preservation of the parent vessel.

Recently, clinical and morphological results have been reported, as well as technique related complications, outlining differences between institutions referral patterns and indications [6,18,24].

The purpose of the present paper is to report and analyse our experience of AA treatment in Bicêtre since we started to use GDC in 1993. Comparison of our data with those available in the literature aims to look for general guidelines to be proposed for the endovascular management of these lesions.

Materials and methods

Clinical Data

During the first year of GDC use in Bicêtre in 1993 we restricted our indications to aneurysms that were felt to be non-surgical [19]. Since then, under an agreement established between our neurosurgical group and our team, all intradural AA felt to be both surgical and embolisable, have been embolised. Appropriate vascular disposition for a safe endovascular approach [4], a clear "working projection" for optimal visualisation of the neck of the AA, appropriate AA size to neck ratio, and AA size to parent artery diameter ratio [19] were among the criteria used to favor embolisation vis-à-vis a surgical approach.

Ruptured and non ruptured AA are included in this series.

For ruptured AA, we try to treat the lesions as rapidly as possible after the diagnosis of SAH, ideally within three days after the onset of the haemorrhagic stroke. Whenever possible, the treatment takes place at the same time as diagnostic angiography, as a continuation of the same procedure. Ruptured aneurysms were considered appropriate for embolisation according to the previously described criteria, but independently from the grade on admission. Surgery was performed if the patient was considered to be a poor candidate for embolisa-

tion, or if the attempted endovascular procedure failed.

Size, shape, multifocality, distribution within a possible surgical field, neurological status, and family history were taken into consideration in the indications for unruptured aneurysms.

Giant, traumatic, mycotic, or dissecting AA were not initially considered for primary endovascular treatment, at least as far as the occlusion of the lesion with preservation of the parent artery was concerned. They are not included and will not be discussed in this paper.

In all patients, follow-up controls included general and neurological examination. Clinical evaluation was performed with both the classical Glasgow Outcome Scale (GOS) and the Karnovsky score. The latter, although designed initially for patients suffering from brain tumours, seemed interesting to us because it allows more precise evaluation of quality of life in patients with good GOS.

A Karnovsky score of 100 indicates normal activity without complaints; 90: normal activity with minor signs; 80: normal activity with effort; 70: cares for himself/herself. Unable to carry on normal activity or to do active work; 60: requires occasional assistance; 50: requires considerable assistance and frequent medical care; 40: disabled. Requires special care and assistance; 30: severely disabled. Hospitalisation required; 20: very sick. Hospitalisation necessary; 10: moribund; 0: dead.

In all cases, follow-up angiography was performed at three months and one year after the initial procedure. When the AA was considered to be totally occluded, controls at three years were performed. Intermediate controls at six and 18 months were proposed in cases of partial occlusion of the AA and/or if recanalisation or regrowth of the lesion was demonstrated. If needed, the AA was again embolized with GDC during one of these sessions. We assessed the rate of occlusion as follows: complete angiographic exclusion of the sac was considered a 100% occlusion, a persistent orifice demonstrated on one or two projections was equivalent to 90-99% and 80-89% occlusion respectively, while partial opacification of the sac corresponds to a less than 80% disconnection.

Technical Aspects

Since September 1998, 3D angiography has been routinely performed (GE Advantx LCN+, SUN workstation) for evaluation of all aneurysms. This allows a very precise delineation of the AA and optimal analysis of its neck. Architecture of the ruptured AA is analysed to find the precise rupture point of the AA, indicated by a false - or pseudo-aneurysm.

All procedures are performed under general anaesthesia. After femoral puncture at the groin, a 6F Headway guiding catheter (Nycomed Amersham, Paris, France) is positioned in the main arterial trunk harbouring the aneurysm (internal carotid or vertebral artery) and the patient is given a bolus of intravenous heparin (50 IU/kg) followed by an infusion to maintain full heparinization (500-1000 IU/h). For ruptured AA treated at the acute stage, heparin is preferentially started after deployment of the first coil in the AA cavity.

No direct carotid puncture was performed in this series.

The lesion is approached under fluoroscopy and road-mapping with a Tracker microcatheter (Boston Therapeutics, Fremont, Calif) mounted on a Dasher microguide (idem). All ruptured cases are embolized with GDC 10 coils placed through a tracker 10 system. Tracker 18 and GDC 18 are used for unruptured AA which are larger than 8 mm; if smaller, GDC 10 are preferentially used.

Continuous coaxial saline solution flushing (without heparin) into the guiding catheter and microcatheter is used. Dense packing is at-

tempted in every case. We avoid "overpacking" ruptured AA, as it is clearly associated with intra-operative rupture and secondary migration of coils into the false sac; in addition it is our experience that secondary induced thrombosis can occur even without overpacking. We did not use the "remodelling technique"[21] in the ruptured cases of this series.

Full heparinization is maintained for 24h, with the activated clotted time at a level twofold higher than normal in order to avoid clotting complications in the parent artery. The introducer sheath is left in place during this time period and removed after the discontinuation of heparinization and normalisation of the clotting time.

All patients with ruptured aneurysms are treated in the acute phase with calcium-channel blockers. In a few cases, steroids were given

Table 1 **AA treated by GDC: patient population**

	BICETRE	UCLA*	FOR**
Number of pts treated/intended	203	—	208
Number of AA treated/intended	225	—	236
Number of pts treated	180	100	182
Number of AA treated	201	104	203

* UCLA = data of the University of California- Los Angeles reported in reference 18; ** FOR = data of the Fondation Ophtalmologie de Rothschild reported in reference 6.

Table 2 **AA treated by GDC: patient population**

	BICETRE n=180	UCLA n=100	FOR n=208
Ruptured aneurysms	118 (66%)	53 (53%)	150 (72%)
H&H 1	25 (21%)	...	92 (61%)
H&H 2	60 (51%)	36 (68%) (H1+H2)	26 (17%)
H&H 3	20 (17%)	8 (15%)	14 (9%)
H&H 4,5	13 (11%)	9 (17%)	4 (3%)
Unruptured aneurysms	62 (34%)	47 (47%)	58 (28%)

Table 3A **Location of aneurysms treated**

	BICETRE n = 201	UCLA n = 104	FOR (n = 236)
Anterior location:	*148 (74%)*	*53 (51%)*	*189 (80%)*
ICA	90 (45%)	42 (40%)	78 (33%)
MCA	10 (5%)	6 (6%)	49 (21%)
ACA	48 (24%)	6 (6%)	62 (27%)
Posterior location:	*53 (26%)*	*51 (49%)*	*47 (20%)*
Vertebral	6 (3%)	4 (4%)	—
V-B junction	5 (3%)	6 (6%)	—
Basilar	42 (20%)	41 (40%)	—

when increased mass effect from the coils was anticipated.

At day 1, plain X-ray films of the skull are obtained to document the position and stability

Table 3B **Location of aneurysms treated (details)**

Location	No of aneurysms
• carotid-ophthalmic	26
• internal carotid artery cave	6
• supracavernous carotid artery	17
• posterior communicating artery	25
• internal carotid bifurcation	16
• anterior communicating artery	47
• pericallosal artery	1
• middle cerebral artery	10
• basilar apex	28
• basilar trunk	7
• vertebrobasilar junction	5
• posterior inferior cerebellar artery	5
• superior cerebellar artery	4
• P1 segment of posterior cerebral artery	2
• anterior inferior cerebellar artery	1
• intradural vertebral artery	1
Total	**201**

of the coils. After heparin has been stopped, anti-aggregant therapy (aspirin 250 mg/day) is initiated and continued until the first angiographic control study.

Results

Between January 1993 and September 1998, 203 patients (with 225 intracranial berry aneurysms) were treated by GDC embolisation (table 1). There were 72 males (35.5%) and 131 females (64.5%); their mean age was 44.3 years. (0.3-77 years). Seven children below 15 years of age are included in this series, most of them with ruptured AA.

The distribution of ruptured and non-ruptured AA is shown in table 2 (118 patients with ruptured AA (65.6%); 62 with unruptured AA (34,4%)), as well as the Hunt & Hess grading of the patients treated for ruptured AA.

The location of treated AA is summarized in table 3 A and B. The aneurysm size is described in table 4.

Attempted treatment was successful in 180 (89%) of these 203 patients. In the other 11% of cases we were not able to selectively approach the AA due to: access problems (anatomical situation (7 AA), atheroma (2 AA), vascular tortuosity (3 AA), spasm (1AA)), or failure to deliver stable embolic devices (large orifice (11 AA)). These latter failures occurred at the beginning of our experience pointing to inappropriate patient selection or non-availability of the so-called "remodelling technique"[21].

In this population of 180 patients, 124 had a single aneurysm, 37 had two, 17 had three and two had four or more aneurysms. Thirteen patients presented with "mirror type" AA that were considered as metameric lesions 3. We actually embolized 201 (89%) of the 225 berry AA diagnosed.

The number of procedures required to satisfactorily exclude the AA is summarized in table 5. Immediately after embolisation 100% occlusion was achieved in 58 aneurysms, 90-99% occlusion in 97 aneurysms, 80-89% in 35 aneurysms and less than 80% in 11 aneurysms.

Transient technical complications occurred in 11.6% of cases (tables 6 and 7). Since we began strictly applying our anti-coagulation protocol, these complications have nearly disappeared.

Table 4 **Aneurysm size**

	BICETRE n=201	UCLA n=104	FOR n=203
<11 mm :	173 (86%)		
11-15 mm :	21 (10%)	50 (48%) <15mm	203 (100%) <15mm
16-25 mm :	7 (4%)	29 (28%)	0 (0%)
>25 mm :	0 (0%)	25 (24%)	0 (0%)

Complications leading to permanent deficits occurred in 3.1% of cases: they are summarized in table 7.

From the nine *embolic complications* that were seen (5%), one was asymptomatic and did not require treatment; eight were eloquent and necessitated medical therapy (five heparin treatment, three fibrinolysis with urokinase): four patients recovered totally and four have persisting deficits. In two other patients, what was believed to be *electric shocks* on surround-

Table 5 **Number of procedures per patient**

	BICETRE n=201	UCLA n=100	FOR n=160 (f.up)
1 procedure	172 (86%)	73 (73%)	141 (88%)
2 procedures	23 (11%)	18 (18%)	18 (11%)
3 procedures	4 (2%)	8 (8%)	1 (0.6%)
4 procedures	2 (1%)	1 (1%)	0 (0%)

Table 6 **Complications of GDC treatment in rupture and unruptured AA**

	BICETRE	Coop. Study (*)	FOR
Silent or transient	23 / 180 (12%)	—	—
Permanent	6 / 180 (3%)	37 / 403 (9,2%)	11 / 182 (6%)
(*) Ref. 8			

Table 7 **Complications of GDC treatment in ruptured and unruptured AA: details of technical problems and related morbidity**

	Prolapsed GDC 1 loop	2-3 loops	emboli	rupture of AA	Induced spasm	others	total
Silent	4	6	1	4*	4	0	19
Eloquent	0	0	8	—	0	2**	10
• Med.Trtmt	0	0	(5)	—	0	0	(5)
• Fibrinolysis	—	—	(3)	—	—	—	(3)
Persist.	0	0	4	—	0	2	6/29
Total	4	6	9	4	4	2	29

* includes 1 case, that was ruptured during diagnostic angio; ** 2 electric shocks: 2nd nerve (horizontal hemianopsia); and brain stem perforators (mesencephalic stroke during 4th session with GDC 18 of 1st generation)

Table 8 **Morphological follow up of GDC treated cases (angiographic control at 1 year minimum)**

	BICETRE n = 158	FOR n = 152
100%	96 (61%)	123 (81%)
90-99%	36 (23%)	26 (17%)
80-90%	21 (13%)	
<80%	5 (3%)	3 (2%)

ing nervous structures lead to neurological complications. These occurred with first generation GDC that necessitated a detachment time exceeding 15 minutes: in one case of a large carotid-ophthalmic aneurysm densely packed, the patient presented with a horizontal hemianopsia[19]. In another patient a large basilar tip aneurysm pointing in the posterior perforated space needed to be coiled in several sessions. During a long detachment time with the last coil spasm occurred on both P1 segments and perforating arteries, without any coil displacement, leading to a mesencephalic stroke. Such complications have not occurred since the introduction of the latest generation GDC.

As a procedure related complication, a 75 year old female (ruptured non-surgical large neck anterior communicating AA in Hunt and Hess grade IV) died from haemorrhagic infarction after fibrinolysis for a thromboembolic event.

Protrusion of GDC loop(s) in the parent artery lumen never led to embolic complica-

Table 9 **Clinical results of GDC treated cases**

	Mortality		
	BICETRE	UCLA	FOR
Unruptured	0/62 (0%)	4/40 (10%)	1/50 (2%)
Ruptured	13/118 (11%)	7/48 (16%)	15/132 (11%)
HH 1,2	3/85 (4%)	0/32 (0%)	11/118 (9%)
HH 3-5	10/33 (30%)	7/16 (44%)	4/14 (29%)

Table 10 **Clinical results of GDC treated cases: mortality (details)**

	BICETRE	UCLA	FOR
Patients with			
Ruptured aneurysms	*n = 118*	*n = 48*	*n = 132*
periprocedural	3 (2,5%)	—	4 (3%)
due to re-Hemorrhage	1 (0,8%)*	—	1 (0,7%)
SAH complication	7 (5,9%)	—	8 (6%)
ICU complication	4 (3,9%)	—	2 (2%)
Total	*13 (11%)*	*7 (15%)*	*15 (11%)*
* refused complementary surgery			
Patients with			
Unruptured aneurysms	n = 62	n = 40	n = 58
	0 (0%)	*4 (10%)*	*1 (0.5%)*

tions; these patients were treated by the same anticoagulation protocol previously described and did not necessitate coil removal. No symptom was noted during the follow-up period and no secondary occlusion of the parent vessel occurred.

No secondary coil migration has been noted in this series. *Vasospasm* induced during the procedure (2%) never led to any clinical problem (except for the particular case described above). *Rupture of the aneurysm* occurred in four cases (one of them during diagnostic angiography before any therapeutic attempt). Intraprocedural rupture (2.5%) occurred in AA treated in the acute phase after SAH. Treatment was completed in all of these patients despite the rupture. Three of these four cases had an excellent outcome (GOS 1; Karnovsky score of 100 in two cases, 90 in one case), while one died from the severity of the initial SAH with Hunt and Hess grade 5.

Follow-up Results

Results are separated into morphological and clinical data.

Morphological follow-up (table 8) could be obtained for 158 of the 201 aneurysms. Clinical follow-up was not possible in 11 patients because of poor outcome; follow-up angiography could not be obtained in 17 cases. Thirteen cases were embolised recently and are awaiting control angiograms.

Total (100%) occlusion was achieved in 96 aneurysms (60.8%) at follow-up, 90-99% in 36 aneurysms (22.8%), 80-89% in 21 aneurysms (13.3%). Thus 83.6% of the AA treated had total or subtotal occlusion (table 8). Five cases (3.1%) had less than 80% occlusion; all of them were large lesions. For three of these patients secondary parent artery occlusion was recommended because of recanalisation or growth of their large AA; two patients refused to be treated. One of them rebled and died three months later (see below).

The clinical results after GDC treatment of ruptured and unruptured cases are summarized in tables 9, 10, 11 and 12 . The *mortality* was 0% in the group of non ruptured AA, and 11% in the ruptured AA population (table 10). For the most part these numbers do not reflect mortality due to the technique but rather to the disease itself and to its clinical consequences: mortality increases dramatically in the poor Hunt and Hess grades whereas it remains low (4%) in the Hunt and Hess grades 1 or 2 (table 9).

The overall results of our management of ruptured AA show a good Glasgow outcome in 85.6% of patients (table 11). Poor results (GOS 3 and 4) were only seen in 3,4 % of our patients with ruptured AA; these correspond mostly to patients in bad initial neurological conditions (Hunt and Hess 3-5). If we analyze the clinical outcome according to the Karnovsky score (table12), the quality of life of the embolized patients at long term follow-up is very satisfactory with in most cases no or minimal symptoms, if the patients were initially in good Hunt and Hess grades or if their clinical and neurological pre-operative status was satisfactory. This reflects the safety of our management with GDC.

Re-bleeding

One patient, as already mentioned, rebled (0.6%) (see above). This particular case excepted, none of our patients suffered haemorrhagic stroke after having the aneurysm coiled , either in the unruptured or in the ruptured group.

Clinical results of unruptured aneurysms in patients with a history of subarachnoid hemorrhage from an additional aneurysm represent a distinct group that deserves separate analysis. Twenty-four aneurysms were embolised in 23 cases with a history of subarachnoid haemorrhage from another aneurysm. The pre-operative Karnovsky score was good (80-100) in 19 patients and mean (80 or less) in four patients. In the post operative follow-up, only one patient had a transient neurological deficit. There was neither permanent morbidity nor mortality in this group. No patient bled, and no patient worsened after treatment.

Discussion

Guglielmi reported in 1991 and 1992 the early clinical experience of detachable platinum microcoils in aneurysms [4,5,10]; the results of the first large series of GDC embolisation have been reported for aneurysms in patients that were initially considered to be poor surgical candidates [8,9,12,13,17,20].

We have tried to compare our results to

Table 11 **Clinical results at discharge in patients with ruptured AA treated by GDC**

	GOS 1-2			GOS 3-4			GOS 5		
	Bicêtre	UCLA	FOR	Bicetre	UCLA	FOR	Bicetre	UCLA	FOR
H&H1	25		83	0		0	0		6
2	57	29	12	0	3	0	2	0	5
3	13	6	7	1	1	0	7	0	4
4,5	6	0	3	3	2	0	4	7	0
Total	*101*	*35*	*105*	*4*	*6*	*0*	*13*	*7*	*15*
	(118)	(53)	(150)	(118)	(53)	(150)	(118)	(53)	(150)
	(85,6%)	(66%)	(70%)	(3,4%)	(11,3%)	(0%)	(11%)	(13,2%)	(10%)

GOS 1-2 = good recovery / moderate deficit; GOS 3-4 = severe deficit / permanent vegetative state; GOS 5 = Death

those of two other main series (from the Foundation Ophtalmologique Rothschild (FOR) [6] and the UCLA group [18]) as we share similar approaches and comparable analysis of results. These data have been included in tables 1 to 5 and 7 to 9. Other larger series can be found in the literature [1,14,23,24], but they could not be used for comparison because they did provide enough detailed clinical or morphological data.

Some large neck lesions have either been treated by surgery, or considered for embolisation with "remodelling". Large series detailing this procedure can be found in the literature, analysing both technical data and results [6,21]. We have used this technique in four patients (2% of all the embolised cases); in all the other cases we could manipulate the GDCs to pack the AA without any balloon.

This series represents the type of treatment

Table 12 **Clinical outcome (according to Karnovsky score) at follow-up of patients with AA treated by GDC**

			Karnovski score					
			100	90	70-80	30-60	10-20	00
Ruptured (n = 118)	**H & H**	**Nb of pts**						
AA	1	25	15	10	0	0	0	0
	2	59	33	20	5	0	0	1
	3	21	5	5	3	1	0	7
	4,5	13	2	1	3	3	0	4
	Initial KS	**Nb of pts**						
	100	19	18	1	0	0	0	0
	90	36	14	21	1	0	0	0
	70-80	6	1	1	3	1	0	0
	30-60	1	0	0	0	1	0	0
	10-20	0	0	0	0	0	0	0

and results that can currently be obtained with endovascular management of berry AA. Patient selection is also based on 3D angiography that proves to be of great help for evaluation of these lesions.

Our series shows a good outcome in 85.6% of ruptured AA, with no or minimal residual symptoms (mostly headaches) in about 76% of cases, allowing normal personal or professional activity. The 3.4% with poor outcome had a poor initial clinical state (Hunt and Hess grades 3-5) and thus relate to the initial severity of the haemorrhage and not to technical management of the aneurysm itself.

The low rate of complication reported in this series reflects the way we analyse the angioarchitecture of the selected AA and in particular ruptured ones. It is essential to precisely recognize the false aneurysm that represents the rupture point of the sac. If the ruptured aneurysm is bilobed, we consider a priori the most distal chamber of the lesion to be a false aneurysm. The high rate of intra operative ruptures sometimes reported [2] may reflect a suboptimal anatomic analysis. Our strategy seems to contradict others which advocate dense packing as the only way to secure the morphological result and subsequently the clinical one [6]. Some teams even systematically sacrifice a last coil if it cannot be placed inside the sac, as they always try to fill the AA as much as possible.

We do not perform partial treatment but we avoid overpacking of AA, as this might not only represent a danger but also is shown often to be unnecessary as about 30-40% have subsequent spontaneous thrombosis inside the ruptured AA. This observation has been made by other teams (L. Picard, M. Mawad, personal communication). The postoperative re-haemorrhage rate of the coiled AA is identical in our group and in those promoting overdense packing of the sac. We experienced only one post-coil re-bleeding in our series in a patient who refused complementary treatment: this case proved to be related to aneurysm growth and not coil compaction or recanalization of the AA lumen. If we exclude that unique case, no bleed or rebleed has occurred in our series.

Treatment of non-ruptured AA seems to be challenged by the data collected by the international study of unruptured aneurysms investigators [16]. The spontaneous risk of haemorrhage is considered as low as 0.05% / year for AA smaller than 10 mm without any history of SAH, and about 11 times higher in patients who had a history of SAH from a different aneurysm that was repaired successfully. The quality of treatment that is offered must optimally carry lower morbidity than the natural history of the disease with no mortality. Some teams actually no longer recommend actively treating unruptured AA smaller than 10 mm [15] and prefer to follow them initially by MRA. Our results are acceptable with regard to that challenge, provided that our patient selection still allows us to recognize the AA that we can safely treat.

Conclusions

In conclusion, our series shows, as already proven by other teams [1,5,6,18,19,22,23,24], that coiling an AA with GDC is an acceptable method of treatment to be proposed to patients with ruptured and unruptured AA. The low morbidity-mortality and absence of rebleeding complications after coiling in our series is probably due to patient selection and architectural analysis. Dense coil packing of AA gives the same result as maximal packing.

New challenges raised by the low risks involved in the natural history of unruptured aneurysms require us to be better than the natural history, and not just the best technician of coil or clip management. Recognition of higher risk subgroups and longer follow-up are needed to assess the validity of our patient selection over time.

References

1 Byrne JV, Solin MJ et Al: Five year experience in using coil embolisation for unrptured intracranial aneurysm: outcomes and incidences of late rebleedings. J Neurosurg 90 (4): 656-663, 1999.

2 Byrne JV, Molyneaux AJ et Al: Embolisation of recently ruptured intracranial aneurysms. J Neurol Neurosurg Psychiatry 59 (6): 616-620, 1995.

3 Campos C, Churojana A et Al: Basilar tip aneurysms and basilar tip anatomy. Interventional Neuroradiology 4: 121-125, 1998.

4 Campos C, Churojana A et Al: Multiple intracranial arterial aneurysms: a congenital metameric disease? Review of 113 consecutive patients with 280 AA. Interventional Neuroradiology 4: 293-299, 1998.

5 Casasco A, Aymard A, Gobin YP: Selective endovascular treatment of 71 intracranial aneurysms with platinum coils. J Neurosurg 79: 3-10, 1993.

6 Cognard C, Weil A et Al: Intracranial berry aneurysms: angiographic and clinical results after endovascular treatment. Radiology 206: 499-510, 1998.

7 Fox AJ, Drake CG: Detachable balloon embolisation for intracranial aneurysms. J Neurosurg 73: 157-159, 1990.

8 Gobin YP, Viñuela F, Gurian J: Treatment of large and giant fusiform intracranial aneurysms with Guglielmi Detachable Coils. J Neurosurg 84: 55-62, 1996.

9 Graves V, Strother C, Weir B: Vertebrobasilar junction aneurysms associated with fenestration: treatment with Guglielmi Detachable Coils. Am J Neuroradiol 17: 35-40, 1996.

10 Guglielmi G, Viñuela F, Sepetka I: Electrothrombosis of saccular aneurysms via endovascular approach, part 1. Electrochemical basis, technique and experimental results. J Neurosurg 75: 1-7, 1991.

11 Guglielmi G, Viñuela F, Dion J: Electrothrombosis of saccular aneurysms via endovascular approach. Part 2. Preliminary clincial experience. J Neurosurg 75: 8-14, 1991.

12 Guglielmi G, Viñuela F, Duckwiler G: Endovascular treatment of posterior circulation aneurysms by electrothrombosis using electrically detachable coils. J Neurosurg 77: 515-524, 1992.

13 Gurian J, Martin N, King W: Neurosurgical management of cerebral aneurysms following unsuccessful or incomplete embolisation. J Neurorsurg 83: 843-853, 1995.

14 Higashida R, Halbach V, Dowd C: Intracranial aneurysms: interventional neurovascular treatment with datachable ballons. Results in 215 cases. Radiology 178: 663-670, 1991.

15 Houdard E: Comment on the article "Unruptured intracranial aneurysms- risk of rupture and risks of surgical intervention". Interventional Neuroradiology 5: 75-76, 1999.

16 The Internation Study of Unruptured Intracranial Aneurysms Investigators: Unruptured intracranial aneurysms: risk of rupture and risks of surgical intervention. N Engl J Med 339: 1725-1733, 1998.

17 McDouall, Halbach V, Dowd CF: Endovascular treatment of basilar tip aneurysms using electrolytically detachable coils. J Neurosurg 84: 393-399, 1996.

18 Malisch TW, Guglielmi G, Viñuela F: Intracranial aneurysms treated with the Guglielmi Detachable Coils: Midterm clinical results in a consecutive series of 100 patients. J Neurosurg 87: 176-183, 1997.

19 Martin D, Rodesch G et Al: Preliminary results of embolisation of non-surgical intracranial aneurysms with GD Coils: the first year of their use. Neuroradiology 38: S142-S150, 1996.

20 Massoud T, Guglielmi G, Viñuela F: Endovascular treatment of multiple aneurysms involving the posterior intracranial circulation. Am J Neuroradiol 17: 549-554, 1996.

21 Moret J, Cognard C et Al: The "remodelling technique" in the treatment of wide neck intracranial aneurysms. Angiographic results and clinical follow-up in 56 cases. Interventional Neuroradiology 3: 21-35, 1997.

22 Raymond J, Roy D: Safety and efficacy of endovascular treatment of acutely ruptured aneurysms. Neurosurgery 41: 1235-1245, 1997.

23 Richling B, Gruber A, Bavinski G: GDC system embolisation for brain aneurysms. Location and follow-up. Acta Neurochir (Wien) 134: 177-183, 1995.

24 Viñuela F, Duckwiler G, Mawad M: Guglielmi Detachable Coil embolisation of acute intracranial aneurysms: perioperative anatomical and clinical outcome in 403 patients. J Neurosurg 86: 475-482, 1997.

Pierre Lasjaunias MD, PhD
Service de Neuroradiologie Diagnostique
et Thérapeutique
Hôpital de Bicêtre
78 rue du Général Leclerc
94275 Le Kremlin Bicêtre (France)
E-mail: pierre.lasjaunias@compuserve.com

COMMENT

The article by Fuse et Al detailing the considerable experience of the Bicêtre group in endovascular management of saccular aneurysms with GDC delineates the excellent results that can be obtained by such a team. However, it also raises many questions regarding the numerous levels on which one can assess outcome following treatment for aneurysms. This team has achieved a permanent morbidity of 3.1%, with no mortality among unruptured aneurysms, and only one documented rebleed from a previously ruptured aneurysm in a patient with recanalization who refused the recommended second treatment. The results compare very favourably with those of two other experienced teams[1,7] in terms of complication rate, morphological results, and clinical outcome. However, how "good" these results are depends not on comparison to other groups, but rather on comparison to the best alternative treatment and ultimately to the natural history of the disease (which is particularly pertinent for unruptured aneurysms). To be "excellent" or even "the best" at delivering a given therapy is not sufficient to warrant adopting it if that management strategy still does not produce better outcomes than the disease if untreated. The true natural history is often difficult to determine, since almost all series incorporate some degree of selection bias when determining which patients make up the untreated group. However, the recent NEJM publication on unruptured aneurysms[4], although potentially biased towards a benign natural history, suggests that our therapy may have to carry extremely low risks in order to justify treatment. In addition, within any group of patients there is always inhomogeneity. Patients are frequently categorized according to clinical grade, age, aneurysm size, etc., in order to try to gauge and compare outcomes while in some way accounting, at least qualitatively, for baseline differences in patient populations. However, the traditionally used variables may be insufficient, since a saccular aneurysm in isolation may carry a significantly different natural history or therapeutic risk than one in a patient with multiple aneurysms, mirror aneurysms, or a positive family history, as these may represent very different diseases[5]. This is analogous to the situation in brain AVMs, where the morphologic distinctions are recognized but the significance of subgroups may still not be fully appreciated[6]. Our knowledge of the differences in the way these subgroups respond to therapy, or behave without treatment, remains limited for both disease conditions.

The relevant issues for follow-up were raised shortly after beginning to use coiling therapy as an alternative to surgery: how good is the acute protection after a subarachnoid haemorrhage? How good is the long-term protection from rebleeding? Is complete angiographic obliteration necessary? What duration of clinical follow-up is required to ensure that the risk of haemorrhage has been eliminated? The volume of cases that has been amassed argues that coiling is fairly successful at providing acute protection from recurrent haemorrhage[1,7,10]. The emphasis thus shifted to the long-term outlook, largely because of the proven track record for surgery in this respect. Again, surgical success has been documented not so much by formal, quality studies as by a volume of collective experience over many years. Clinicians and patients continue to worry about coil compaction, aneurysm regrowth, and residual body or neck filling because the significance of these findings remains uncertain. It

is unclear whether coiling fundamentally alters the clinical course of the disease, or whether the risk remains high with any remnant since whatever factors led to the initial haemorrhage are still at work. These issues raise the question as to exactly what method and duration of follow-up are necessary. Does one look at pictures only? If so, then the length of follow-up required to reassure everyone that the aneurysm is secured will correlate with stability of the appearance on angiographic images, and possibly the cycle of renewal of the vessel wall. On the other hand, if clinical follow-up is the appropriate method, then one may come to ignore small neck remnants or changes in coil mass appearance as long as the patient never suffers from a rebleed. The duration of follow-up required for this would necessarily be longer, demanding a certain number of "patient-years" to reassure us that the risk is below some arbitrary, acceptable limit. To accumulate enough experience, one must bear in mind the difference between following many patients for a short duration versus fewer patients for a long time. Although both may yield the same number of patient-years, long-term follow-up is essential, since recurrent bleeding is not a random event. The protection may be good for several years until the vessel has remodeled in an adverse way, at which time there is a renewed risk of subarachnoid haemorrhage.

The degree of packing required for optimal results is as yet unresolved also, and thus terms such as "sufficient" and "overpacking" are emerging to describe the beliefs of a given therapeutic team regarding the ideal. It is clear that incomplete packing beyond a certain point will often lead to recanalization and / or compaction, and thus ongoing risk of haemorrhage [1,2,3,8,9,10,11]. *However, there is a difference of opinion on whether very tight packing to the absolute limit of feasibility (often failing to deploy the final coil which is attempted, or doing so with significant friction and repositioning of the microcatheter within the aneurysm) is necessary. One may imagine that more coils is always better, but this strategy comes at a cost (both financial, but more importantly potential risk to the patient of aneurysm rupture, coil herniation and thrombosis/emboli, etc.). There is, for every aneurysm, a certain stage at which the change in flow within the aneurysm due to a given mass of foreign material will lead to a favourable clinical course with thrombosis and exclusion of the aneurysm from the circulation. Any coil added after this stage incurs additional risk and cost without achieving further benefit. The stage at which this happens may be influenced by a number of factors, including aneurysm size, direction of flow vis-à-vis the aneurysm neck, neck width, and so on. The degree of packing required to achieve this success is one of the many factors which vary among teams, and about which a given group may feel very strongly, yet which has not been systematically studied to determine the best protocol to follow.*

The reports by Fuse et Al and other experienced teams, while definitely encouraging, lead to a host of unanswered questions which require further study. These research efforts will be perpetual, since therapeutic techniques and equipment also evolve. By the time some of these questions about coils have been answered, the nature of endovascular therapy may have changed significantly from that upon which the initial research was based.

References

1 Cognard C, Weil A et Al: Intracranial berry aneurysms: Angiographic and clinical results after endovascular treatment. Radiology 206: 499-510, 1998.
2 Gurian J, Martin N, King W: Neurosurgical management of cerebral aneurysms following unsuccessful or incomplete embolisation. J Neurosurg 83: 843-853, 1995.
3 Horowitz M, Purdy P et Al: Aneurysm retreatment after Guglielmi detachable coil and nondetachable coil embolisation: Report of nine cases and review of the literature. Neurosurgery 44(4): 712-720, 1999.
4 The International Study of Unruptured Intracranial Aneurysms Investigators: Unruptured intracranial aneurysms: Risk of rupture and risks of surgical intervention. NEJM 339: 1725-1733, 1998.
5 Lasjaunias P: From aneurysms to aneurysmal vasculopathies. Interventional Neuroradiology 5: 105-108, 1999.
6 Lasjaunias P: A revised concept of the congenital nature of cerebral arteriovenous malformations. Interventional Neuroradiology 3: 275-281, 1997.
7 Malisch TW, Guglielmi G, Viñuela F: Intracranial aneurysms treated with the Guglielmi detachable coils: Midterm clinical results in a consecutive series of 100 patients. J Neurosurg 87: 176-183, 1997.
8 Martin D, Rodesch G et Al: Preliminary results of embolisation of non-surgical intracranial aneurysms with GD coils: The first year of their use. Neuroradiology 38: S142-150, 1996.
9 Murayama Y, Viñuela F et Al: Endovascular treatment of incidental cerebral aneurysms: Report on 115 cases treated with Guglielmi detachable coils. Interventional Neuroradiology 5 (Suppl 1): 79-81, 1999.
10 Viñuela F, Duckwiler D, Mawad M: Guglielmi detachable coil embolisation of acute intracranial aneurysms: Perioperative anatomical and clinical outcome in 403 patients. J Neurosurg 86: 475-482, 1997.
11 Yoichi A, Murayama Y et Al: Balloon-assisted Guglielmi detachable coiling of wide-necked aneurysms: Part I - Experimental evaluation. Neurosurgery 45(3): 519-530, 1999.

Phillip J. Porter, M.D.
Neurosurgery and Interventional Neuroradiology
The University of Toronto Brain Vascular Malformation Study Group
University Health Network, Western Division
Toronto, Canada